**IN THE SPECIFICATION:**

Please replace paragraph beginning at page 65, line 2, with the following rewritten paragraph:

*Plasmid construction* – Full length human MINK3 was cloned into pCI (Promega) derived expression vector pYCI under the control of the CMV promoter with an HA epitope tag (AYPYDVPDYA (SEQ ID NO:7)) inserted on the N-terminus by PCR. A kinase mutant form of MINK3 was constructed using the QuikChange mutagenesis kit (Stratagene) with Oligos AGCTTGCAGCCATCAGGGTTATGGATGTCAC (SEQ ID NO:8) and GTGACATCCATAACCTTGATGGCTGCAAGCT (SEQ ID NO:9) to change the highly conserved lysine 54 in the kinase domain to arginine. Full length human NCK was similarly cloned into pYCI with a FLAG epitope tag at the N-terminus. Myc-JNK2 and Myc-ERK1 were constructed in the pCR3.1 vector with a Myc epitope tag (ASMEQKLISEEDLN (SEQ ID NO:10)) inserted on the N-terminus of JNK2 and ERK1, respectively. All the truncation mutants were constructed by PCR.

A1

Paragraph beginning at page 67, line 34, has been amended as follows:

NIK was cloned by its ability to interact with the adapter protein NCK. It associated with NCK SH3 domains via two PxxPxR sequences in the intermediate domain, PCPPSR (aa 574-579; SEQ ID NO:11) and PRVPVR (aa 611-616; SEQ ID NO:12). Both sequences were required for efficient interaction (Su, et al., EMBO J., 16:1279-1290 (1997)). Similar to NIK, MINK3 also interacted with NCK via the intermediate domain. However, PCPPSR is not conserved in MINK3. Instead, MINK3 contained two other PxxPxR sequences, PNLPPR (aa 562-567; SEQ ID NO:13) and PPLPTR (aa 647-652; SEQ ID NO:14), in addition to the conserved PKVPQR (aa 670-675; SEQ ID NO:15). MINK3 likely interacted with NCK through the cooperative interaction with these three PxxPxR sequences. NCK is an adapter protein involved in many growth factor receptor mediated signal transduction pathways (McCarthy, Bioessays, 20:913-921 (1998)). It has been proposed that the NIK-NCK interaction may recruit NIK to receptor or non-receptor tyrosine

A2